THE DENTAL PRACTITIONER

AND DENTAL RECORD

Including the Transactions of the British Society for the Study of Orthodontics, and the official reports of the British Society of Periodontology, the Glasgow Odontological Society, the Liverpool and District Odontological Society, the North Staffordshire Society of Dental Surgeons, the Odonto-chirurgical Society of Scotland, and the Dental and Medical Society for the Study of Hypnosis

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THE DENTAL PRACTITIONER
AND DENTAL RECORD

Vol. X, No. 11 July, 1960

EDITORIAL

FIXING FACES

In this issue of THE DENTAL PRACTITIONER there appears the first part of a paper reviewing present-day treatment of congenital malformations of the facial structures in the young. Whatever the dentist can do to allay the anxiety of the parents and to co-operate with the surgical team responsible for treatment, he is serving not only the patient but also the community at large.

The appearance of the newborn baby afflicted with a facial cleft is grotesque and a dreadful shock for the parents. Unless surgical repair is undertaken the child itself soon suffers severe mental and physical embarrassment from appearance and speech. However, in these cases, the disturbances of growth are often more widespread than appears at first sight, and surgery, while improving certain aspects of the condition, often produces further deviations from the normal pattern of development. The problems posed by these patients are therefore complex and many specialties are involved in their solution.

Although some technical advances have been made and a few new operative procedures suggested, the main surgical thought during the last few years has been centred on a policy of consolidation. Such long-term "follow-up" of patients has helped the surgeon to crystallize his ideas on the most suitable procedure for a particular condition.

Any dental surgeon who has to co-operate in the treatment of these patients must be conversant with both the aims and the limitations of the particular repair which is to be attempted. In the past the dentist's role has been mainly in support of the surgical attack, but in recent years exciting new developments in orthodontic treatment—both before and after operation—have widened the scope of the dentist in this field.

As distressing as congenital defects in children is the increase in the number of patients seriously injured in road accidents. Many suffer facial injuries and here there is the psychological as well as the surgical aspect to consider. Nothing boosts the morale of these patients more than having their faces attended to at an early stage, and the assurance which they can then be given that they will "look all right when it has healed".

In looking back over the last twenty years it is realized that the impetus given to oral surgery by the close wartime association between dentists and their surgical colleagues has not slackened, and since then the creation of many registrar posts has enabled those who benefited from wartime experiences to pass on their knowledge. A glance at dental literature shows, in fact, a gradual broadening of the scope of oral surgery and a steady increase in the technical abilities of the operating dentist.

It is hoped that these articles in THE DENTAL PRACTITIONER may stimulate students to interest themselves in this work and afford a review of the subject for the general practitioner.

CONGENITAL MALFORMATIONS OF THE MOUTH AND JAWS

PART 1

By **R. T. ROUTLEDGE**, F.R.C.S.
Department of Plastic and Jaw Surgery, Frenchay Hospital, Bristol

EMBRYOLOGY

THE development of the face and jaws is a complex process, but a brief reference to it is of importance in acquiring a proper perspective of the commoner congenital abnormalities. It will serve our purpose quite adequately to

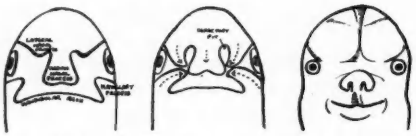


Fig. 1.—Diagrammatic representation of development of face.

streamline the complicated sequence of events, and consider briefly the fate of the nasal processes and the mandibular arch. In the fifth to sixth week of intra-uterine life the mesenchymal layer separating the forebrain and the epithelial roof of the mouth thickens, and grows caudally to constitute the frontonasal processes. Ectodermal thickenings appear on each side of this and become depressed to form the olfactory pits, so dividing the frontonasal process into a median process and two lateral nasal processes. At the same time a triangular-shaped enlargement, called the maxillary process, grows ventrally from the dorsal end of the mandibular arch on each side to fuse with the lateral nasal process along a groove termed the naso-optic furrow, thereby establishing continuity between the side of the nose and the adjoining cheek. The ectoderm along the line of fusion sinks beneath the surface and is canalized to form the nasolacrimal duct.

The apex of the triangular maxillary process continues to grow towards the midline to meet and fuse with the median nasal process to form a definitive upper lip. It should be noted, however, that the mesenchyme of the maxillary processes invades the median area, to make a substantial contribution to the philtrum of the lip. This explains the innervation of the philtrum by the maxillary nerve. (*Fig.* 1.)

In the sixth week, palatine processes grow in from the inner surfaces of the maxillary processes. They are separated, at first, by the bulk of the tongue, but, as the mandibular arch continues to grow, the tongue is carried caudally away from the developing palate so that the two palatine processes may fuse in the midline with each other and with the developing nasal septum.

With so complex a process it is not surprising that congenital malformations occur not uncommonly during the formation of the face and palate.

PARTIAL AGENESIS OF FACIAL SKELETON

For reasons which are, at present, unknown, development of the normal facial skeleton may be retarded. This failure to develop normally

Fig. 2.—Unilateral congenital partial facial agenesis showing not only under-development of the left half of the mandible and the left maxilla, but also of the soft tissues of cheek and chin.

may be bilateral or unilateral, and may vary from the most extreme degree to a deformity so slight as to pass almost unnoticed.

Fig. 3.—Congenital unilateral partial facial agenesis largely confined to an under-development of the right half of the mandible. Note that, at first glance, it might appear to be the left side which is under-developed. This appearance results from a swing to the right due to an unopposed normal growth of the left half of the mandible.

In its most marked form there is an extreme under-development of the maxillæ, the malar zygomatic complexes, and the mandible, associated frequently with malformations of the ears and with facial palsy. The name "Treacher-Collins syndrome" has been given to such cases, and treatment of the condition is difficult and only completed after very many stages.

The commoner deformity is the so-called "unilateral congenital partial facial agenesis", which may involve the mandible only, but which quite frequently involves the maxilla also, and is then often associated with congenital malformations of the ear and a unilateral facial palsy (*Fig.* 2). In the extreme deformities we have noted an absence of the ascending ramus, neck, and condyle of the mandible, and a complete lack of the normal glenoid fossa. This seriously complicates the treatment, for, as a first stage in reconstruction of the lower jaw, some form of bearing surface must be constructed to accommodate the upper end of the bone-graft which will later be inserted to replace the missing segment of mandible. A suitable bearing surface is provided by a small bone-graft inserted as a kind of ledge to bridge the gap between the posterior root of the zygomatic arch and the mastoid process.

Much speculation exists as to the correct age at which to carry out reconstructive procedures on patients suffering from a congenital unilateral partial agenesis of the mandible. Broadly speaking, there are two alternatives:—

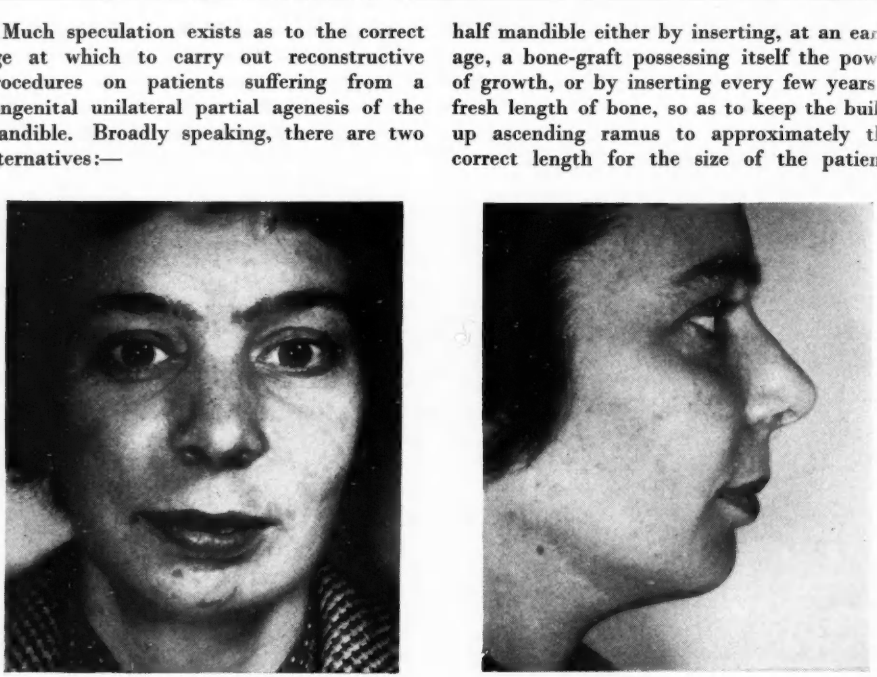


Fig. 4.—Same case as in *Fig.* 3 treated by onlay bone-graft.

1. To await completion of growth in the facial skeleton, accepting all this time an ever-increasing facial deformity and a serious disturbance of dental occlusion, and then to carry out onlay bone-grafting procedures designed purely to camouflage the misshapen jaw. As far as facial appearance is concerned, this technique can give most pleasing results, but an intra-oral examination is sufficient to make any self-respecting orthodontist blush for shame. (*Fig.* 3.) As a result of the failure of one half of the jaw to develop normally, and the consequent deviation of the normal half, both dental arches are seriously disturbed. Yet, even so, occlusion of a kind is achieved and all that we can do is to accept such occlusion as exists, for any attempt to improve on it usually ends in a serious impairment of function. (*Fig.* 4.)

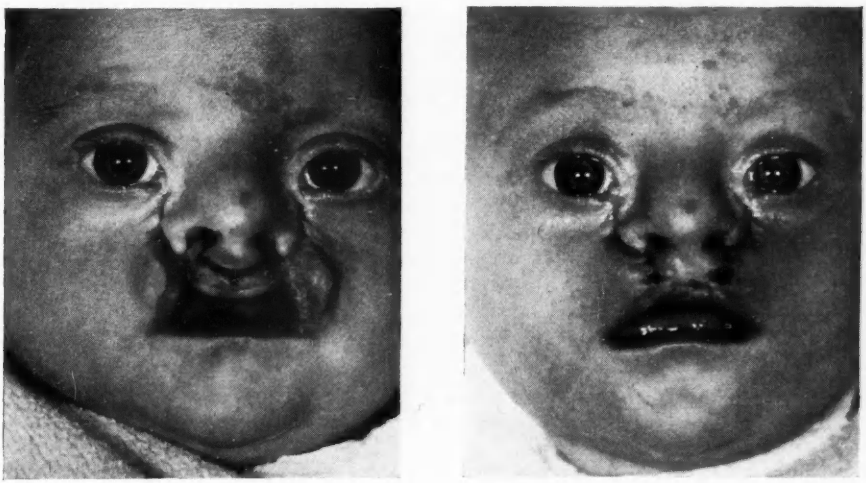
2. The second plan, about which many oral surgeons have become most enthusiastic, is to simulate normal growth of the under-developed half mandible either by inserting, at an early age, a bone-graft possessing itself the power of growth, or by inserting every few years a fresh length of bone, so as to keep the built-up ascending ramus to approximately the correct length for the size of the patient. Continuous orthodontic treatment has to be provided in these cases, in order to adjust the teeth to the repeated "growth" spurts.

A good deal of work has been done on the growing bone-graft in the United States, and one published series reports encouraging results following the grafting of a metatarsal bone, complete with its growth centres, into the ascending ramus of the mandible. Undoubtedly, this is a promising line of research as regards the treatment of a condition which is not uncommon, and which can prove to be both disfiguring and disabling.

MACROSTOMA

A macrostoma, or transverse cheek cleft, is due to a separation in whole, or in part, of the mandibular arch and its maxillary process. It may be unilateral or bilateral, and of any degree, from a slight lengthening of the mouth to a transverse cleft extending across the cheek almost to the ear. Surgical treatment

of the condition is not without its disappointments, as it is virtually impossible to form a normal-looking commissure of the mouth when the orbicularis oris muscle is deficient, as, of course, it is in these patients. Simple paring of the cleft, with a meticulous closure in layers, is the simplest operative procedure.



A B

Fig. 5.—A, Facial cleft. The naso-lacrimal duct is clearly seen extending on each side to the inner canthus of the eye. B, Showing the closure of the bilateral cleft lip. The nasolacrimal furrow remains to be closed.

FACIAL CLEFT

Rarely, the maxillary process fails to fuse with the lateral nasal process. In this event the lip is cleft, and the nasolacrimal duct forms an open furrow along the side of the nose up to the inner canthus of the eye. It is a difficult condition to treat, but an attempt should be made to reconstitute a patent nasolacrimal duct, opening into the nasal cavity below and onto the lower lid margin above. The lip cleft is closed by any one of the standard procedures. (*Fig.* 5.)

CLEFT LIP

A lateral cleft is the commonest congenital defect involving the lips and it is due to a failure of fusion between the maxillary process and the median nasal process. This failure of fusion may be complete or incomplete, unilateral or bilateral.

COMPLETE UNILATERAL CLEFT LIP

The cleft, which may be very wide, extends the full length of the upper lip into the floor of the nostril, which is greatly increased in its transverse diameter. The alveolus is usually cleft and very often there exists a cleft of the palate as well. The maxilla is under-developed on the cleft side, so that the alar base is depressed below the level of its fellow, giving the typical deformity of a wide, flat nostril, with marked deviation of the tip of the nose and the columella. (*Figs.* 6 and 7.)

Treatment.—As a rule, operation is planned for a time when the infant has attained a normal feeding régime, and is about 10 lb. in weight and gaining satisfactorily. This usually means that the child is about three months old when the primary repair is performed. Minor degrees of incomplete cleft lip can safely be left much later than this, but it is important that the more severe clefts are dealt with early because, once the cleft is soundly repaired, the reconstituted muscle-pull of the lip

exerts an important moulding effect on the underlying bony structure. Simple alveolar clefts often close without further treatment once normal tension is obtained in the overlying lip.

It is important to achieve primary wound healing, and to this end a pre-operative hæmoglobin estimation of 80 per cent or more is essential, and nasal cultures should be free from pathogenic organisms.

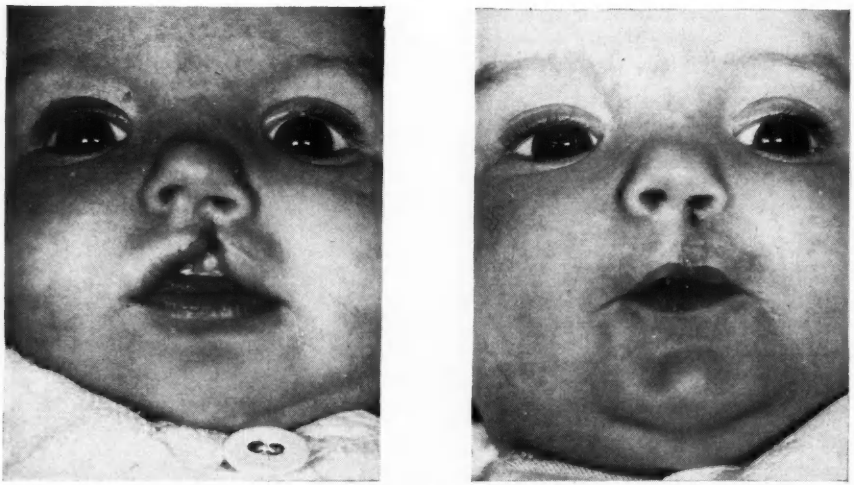


Fig. 6.—Unilateral incomplete cleft lip before and after repair.

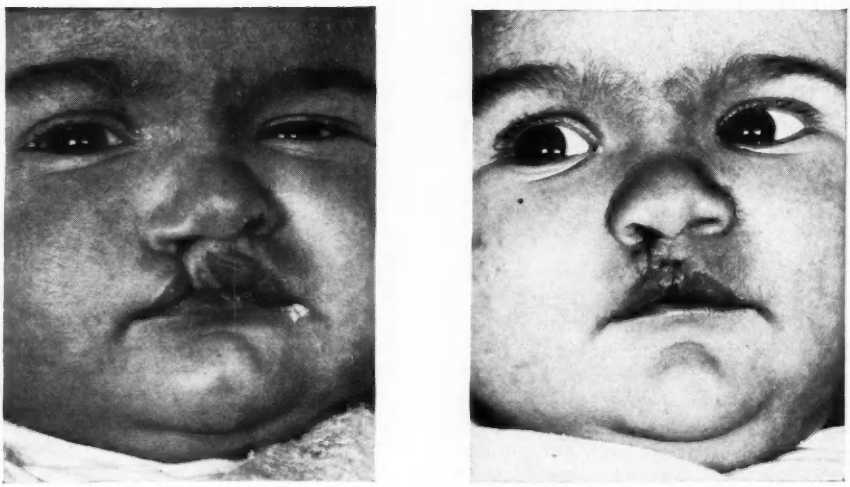


Fig. 7.—Unilateral complete cleft lip before and after repair.

Many operations have been devised for the single cleft, but they all have certain basic features in common. The aim is to produce a symmetrical lip of adequate length, with a scar

that will not contract; to ensure firm and accurate apposition of the muscle-layer of the lip, and to close the floor of the nostril so as to produce as symmetrical a nose as is practicable.

A popular contemporary procedure is the Mesurier type of repair. In this technique

Fig. 8.—Diagrammatic representation of the Mesurier type of cleft lip repair.

adequate length is achieved by cutting transversely into the lip, so as to produce interlocking flaps. (*Fig.* 8.)

It will be seen that the resultant scar is Z-shaped, and this effectively combats any tendency to contraction along its length. When the flaps have been cut they are both extensively undermined right out into the cheeks, until the wound edges can be brought together without tension. Hæmostasis is secured and the wound meticulously repaired in layers, being very certain to achieve sound union of the muscle layer.

No dressing is required, nor is any form of apparatus needed to avoid tension on the wound. The sutures may be removed on the sixth day, and there is no reason why breast-feeding should not be continued throughout, if facilities exist for accommodation of the mother.

BILATERAL CLEFT LIP

In this condition both clefts may be complete, both incomplete, or one complete and one incomplete. In the commoner bilateral complete cleft lip, wide clefts extend into both nostrils, so that the pre-maxilla is attached by a narrow pedicle to the anterior end of the nasal septum. It is often extremely displaced, being rotated through as much as 90°, and in most cases the palate is widely cleft, though occasionally the lateral palatal elements have collapsed inwards behind the projecting pre-maxilla to an extent that makes it impossible to replace it. (*Figs.* 9 and 10.)

Fig. 9.—Bilateral incomplete cleft lip before and after repair.

Treatment.—Operation is undertaken at the same time as for unilateral cleft lips, and the pre-operative standards of fitness are similar.

Again, there exist a host of operations for the treatment of this condition, but here the issues are less clear cut, and differences of

opinion mainly concern the treatment of the pre-maxilla. In the simplest form of operation a straightforward lip repair is performed so that muscle-pull in the reconstituted lip will mould the pre-maxilla into an approximately normal position. The skin covering the pre-maxilla, the so-called pro-labial skin, though it belongs in large part to the columella of the nose, has to be used in this repair to close the large central defect in the lip, even though it means producing an unsightly deformity of the nasal tip which will need secondary correction at a later date. Using this procedure it is not always possible to close both sides of the widest clefts at the one operation, especially when displacement of the pre-maxilla is extreme. (*Fig.* 11.)

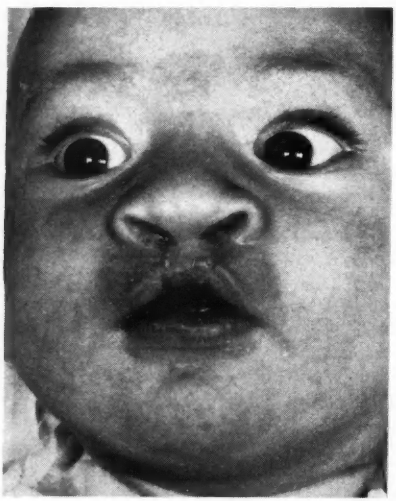


Fig. 10.—Bilateral complete cleft lip before and after repair.

Fig. 11.—Diagrammatic representation of a standard form of double cleft lip repair.

Many techniques have been described which include a direct surgical attack on the pre-maxilla aimed at repositioning it so that the lip may be more easily repaired over it. There seems little doubt, though, that this bony peninsula, suspended from the nasal septum by its narrow pedicle, does not stand up well to surgical trauma, and is prone to undergo a kind of avascular necrosis such as is seen after trauma to the head of the femur. The earlier, somewhat brutal attempts at replacing the pre-maxilla and wiring it into position,

only too often led to its total disappearance and the subsequent collapse of the two lateral maxillary arches with unsightly flattening of the lip and gross disturbance of dental occlusion.

More recently, Denis Browne advised removal of a triangular segment of the nasal septum, just behind the attachment of the pre-maxilla, to permit it to be retroposed, and this procedure may conveniently be combined with the pinning of the pre-maxilla into a corrected position. Yet even this relatively minor degree of trauma may be sufficient, in some cases, to cause a disappearance of the pre-maxilla.

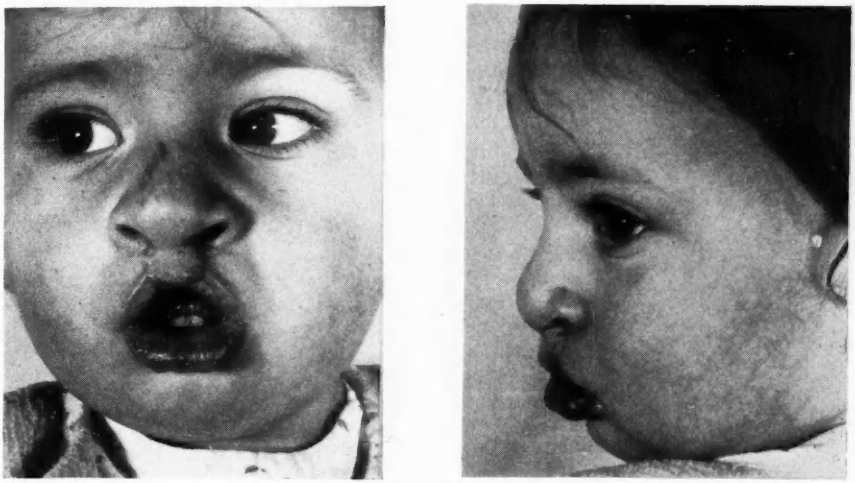


Fig. 12.—After primary repair of double cleft lip, showing short flat upper lip with the tip of the nose tied down by a virtual absence of columella.

As mentioned, the loss of the pre-maxilla gives rise to a most unsightly collapse of the central portion of the upper lip and an upper dental arch, which is virtually triangular in outline, so there is a good deal to be said for the older, but by no means outdated, simple repair of the soft tissues only, which, at any rate, leaves the pre-maxilla unscathed.

Secondary Work on the Repaired Cleft Lip.—In practised hands, the Mesurier type of single cleft lip repair gives uniformly acceptable results, and corrective work carried out at a later date usually involves little more than the adjustment, in some cases, of the red margin of the lip.

Mention has been made, however, of the under-development of the maxilla on the cleft side. This lack of development affects particularly that portion of the maxilla which underlies the lateral attachment of the nostril base, so that the entire nose tip lies askew, with consequent deformation of the alar cartilages. No amount of surgical camouflage can disguise the fact that one nostril lies at a

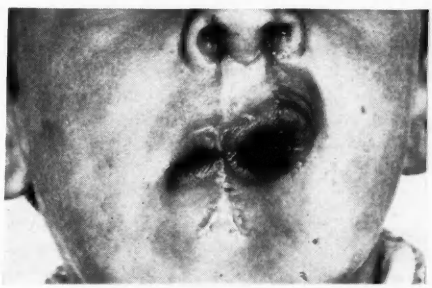


Fig. 13.—The pro-labial skin has been freed and inset to lengthen the columella, and the defect has been filled with a shaped flap, cut from the lower lip, which is nourished through the connecting bridge by the labial artery.

lower level, but encouraging results have been achieved in the correction of this deformity by inserting an onlay bone-graft to the maxilla, introduced into a pocket formed for its reception beneath the alar base. The approach is conveniently made through a small incision in the upper buccal sulcus.

A great deal of corrective work is usually inevitable after a primary repair of complete bilateral cleft lip. The simple truth is that there is never sufficient material in these lips to do more than simply close the gap. At some stage it becomes necessary to introduce more soft tissue into the lip, and there are different ways of doing this. The point was made earlier that the use of the pro-labial skin to close the central defect resulted in an ugly snub-nose deformity, the tip of the nose being tied down and the columella being almost non-existent. This deformity has attracted a good deal of attention, for if it can be successfully corrected the basis of a good lip repair has been established. Advancement of the columella is the key to a successful correction,

Fig. 14.—The connecting bridge has been divided to re-form normal upper and lower lips.

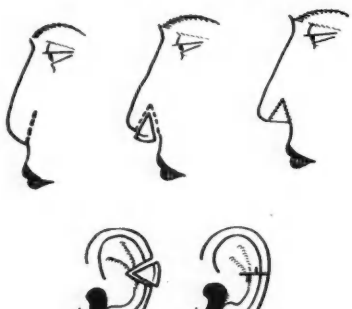


Fig. 16.—Diagrammatic series to illustrate columellar lengthening by means of a composite ear graft.

Fig. 15.—This profile view clearly shows the increased fullness of the upper lip and the improved appearance of the nose tip.

and broadly speaking, this may be carried out in one of two ways.

First, the pro-labial skin may be dissected out of the centre of the lip and the skin over the dorsum of the nose undermined so that the tip of the nose can be drawn up to a normal position, being retained there by suturing the pro-labial skin to the cut edge of septal mucosa, so as to reconstitute a columella of normal length. It will be appreciated that this leaves a central defect in the lip. This may be closed by advancement flaps, by a full thickness free graft, or by means of a cross-lip flap. This latter procedure has been widely used, for, at the expense of a scar in the lower lip, a large defect may be filled and muscle may be introduced to that part of the upper lip which is deficient in it. The accompanying photographs show what an acceptable result can be obtained by such a method. (*Figs.* 12–15.)

Recently, a simpler method altogether of lengthening the short columella and correcting

the snub nose has been receiving a good deal of attention. In this technique a longitudinal incision through the full thickness of the nasal septum permits the tip of the nose to come out to a normal position at the expense of a triangular-shaped defect in the septum. This defect is then repaired by means of a full-thickness chondro-cutaneous graft cut from the ear. (*Fig.* 16.) This method seems particularly suited to the case where a good-looking lip has been obtained at primary repair and only the nose tip needs revising. In cases where the lip repair is not acceptable, the cross-lip flap procedure appears preferable.

[*To be continued*]

The Fractured Tooth-root:
A Systematic Approach to its Removal

The techniques are not original, the approach is, and the subject is discussed under six headings:—

1. Factors responsible for tooth-fracture during extractions: faulty technique; anatomical variations in bone density; pathological changes in roots or bone.

2. Classification of fractured roots: gingival line, mid-root, and apical third fractures.

3. Instruments: root forceps; elevators; bone chisels and burs; artery forceps; probes; scalpels; retractors; lights; and aspirator.

4. Clinical hints: X-ray; thorough examination of the root and surrounding bone; spend ten minutes looking and ten seconds operating; assistant to retract cheek and to aspirate with a fine-nozzled aspirator; use cotton-wool swabs in tweezers with a scrubbing action; raise flap if necessary and nip away bone with rongeurs.

5. Basic technique for root removal:—

a. Root Forceps.—The beaks of the forceps are wedged into the periodontal membrane. This method can be used only for gingival line fractures and where the alveolar crest is thin enough to allow the application of forceps and where the roots are normal.

b. Root Division.—This is most useful for lower molars and the division is carried out with a fissure bur. The separated roots must be normal.

c. Surgical Extractions.—Any root can be removed by this method but it is destructive of bone. The amount of buccal bone to be removed will depend on the class of fracture. Minimum bone removal is required in gingival line fractures and thereafter the exposed root is removed with forceps. For mid-root and apical fractures more bone must be removed and the root extracted with an elevator applied mesially or distally. Apical third root fractures may be dealt with through an "apicectomy window". Palatal roots of posterior teeth may be brought over to the empty buccal socket after first removing the septum of bone.

d. Elevators.—These instruments work on the principles of wedges and levers. The wedge principle is used when a conical root is forced upwards from its socket by forcing an elevator down into the periodontal membrane. A leverage is used for the roots of multi-rooted teeth when the fulcrum is either the buccal plate of bone or the other root. The wheel and axle principle is one where the rotation of the wheel rotates the axle. The Winter's exolever No. 11 or Ploughshare works on this principle. No great force is necessary if the application of the instrument is correct. Indeed, too much force may fracture the bone. Retained roots of lower molars may be removed on this principle.

6. An outline of the systematic approach. The retained root is placed into one of the three classifications mentioned and the method of removal decided upon. Root forceps and division of roots are applicable only to gingival line fractures. Surgical extraction may be used for any root. Elevators working on the wedge principle may be used for gingival line fractures when the roots are not too long and for normal roots in the other two classes. Lever elevators are applicable to all these classes when adequate fulcrum can be obtained. Elevators working on the wheel and axle principle cannot be used for apical third fractures because the roots are too deeply placed in the bone.—SUTHERLAND, K. J. G. (1960), *Aust. dent. J.*, **5**, 1.

PRE-CLINICAL TEACHING— THE PHANTOM HEAD

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SINCE a pre-requisite to the training of dental students in operative procedures and instrumentation demands the use of a phantom head or manikin, it is of considerable importance that the "neck" movements of the manikin simulate as much as possible those of a patient in the dental chair. Failure to provide natural movement during the training period creates psychological and technical difficulties for the student when he is introduced to clinical dentistry.

Fig. 1.—The conventional type phantom head with a hinge-joint giving one plane movement.

Fig. 2.—Side view of the modified phantom head.

Myers and Lawton (1952) stressed the importance of a "pre-clinical unit" approach to phantom head work. Emphasis was laid on the necessity for discipline in the placing of instruments, dental engine, and lights, to create conditions simulating those found at the chairside. They, however, illustrated a phantom head with a central hinge-joint which fails to provide all the head positions clinically experienced.

Peterson (1959) admits that, as a patient's movements cannot be duplicated entirely on a phantom head, some oral hygiene techniques have to be modified on transference to the patient and that this is not entirely satisfactory. He illustrates, like Myers and Lawton, a phantom head with a central hinge-joint, and from visits to dental schools in this country and in the United States it appears that this is the type in common use.

Direct and indirect vision is employed in the use of both hand and rotary instruments, but in order to facilitate more accurate working, a direct approach should be adopted wherever possible. With the introduction of high-speed rotary instruments which cut the tooth tissues with little or no guiding torque, such an

approach becomes almost a *sine qua non*. Students will require pre-clinical instruction on high-speed cutting, and direct vision can only be procured on the "maxilla" if the phantom head provides a side and tilting movement as well as a backward one. To approach the buccal and palatal surfaces of maxillary teeth it is of considerable help if the patient's head can be turned towards or away from the operator, so the student must have pre-clinical instruction on positioning himself and the phantom head, in order that he can work with optimum comfort and accuracy at the chairside.

Until recently the phantom heads in the Edinburgh Dental School were of the conventional hinge type (*Fig.* 1), but a hinge limits the head movements to one plane. It does not allow any tilting or side movement, as is found in the articulation of the skull to the vertebral column, and although this articulation is not a ball-and-socket one, the rotation which the skull makes with the neck closely resembles that found in ball-and-socket movement.

The one-plane hinge has now been replaced by a ball-and-clamp type joint (*Fig.* 2) through which full anatomical movement is provided. Two important factors must be considered in this modification (*see Fig.* 3):—

1. The "ball" of the joint must be of sufficient diameter to supply adequate frictional grip when the "clamp" is closed on it. The diameter of the ball shown is $1\frac{1}{4}$ in.

2. The rod attaching the head to the ball must be kept within certain dimensions. A rod diameter of $\frac{3}{8}$ in. was regarded by the designing engineers* as the most suitable, since the range of head movement would be restricted by any thicker rod and the strength of the junction jeopardized by a thinner one.

Under clinical conditions, an additional range of movement is provided by the dental chair. Instrumentation from the front of a patient usually requires the chair to be placed in the vertical plane, whereas from the back of the patient the chair is tilted to approximately forty-five degrees. By incorporating on the phantom head support-bracket (*Fig.* 3) two fixed positions, A in the vertical plane and A_1 at 45°, these extremes of chair movement are duplicated.

It is in the pre-clinical training of the student that the foundations of good or bad

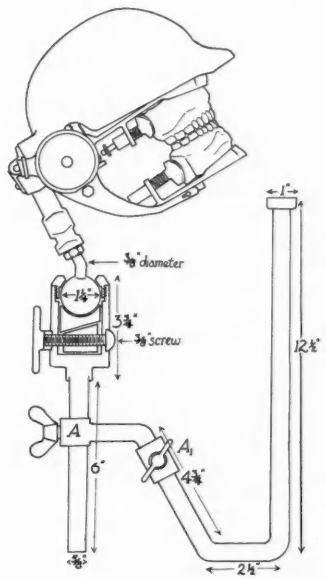

Fig. 3.—Scale drawing showing the construction of the "ball-and-socket" joint with the alternative settings A and Al in the support bracket representing the movements of the dental chair.

instrumentation are laid. The described modifications in the phantom head, along with the dual positioning in the bench support bracket, provide the teacher and the student with a manikin which can reproduce the chairside positions of a patient.

Acknowledgements.—For the diagram I wish to thank Miss Mary Benstead, medical artist, and for the manikin photographs Mr. A. Hunter, M.B.E., and Mr. D. Standen.

* Charles Henshaw & Sons Ltd., Architectural Metal Workers, Russell Road, Edinburgh.

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ORAL MANIFESTATIONS OF BLOOD DISEASES*

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THE privilege of addressing this Society affords me the opportunity to emphasize the value of ready liaison between dental medicine and the clinical laboratory, particularly as it affects the specialty of hæmatology. The Royal Dental Hospital and its School form one unit of the St. George's Group of teaching hospitals, and during the past few years there has developed a close, and, I believe, rewarding liaison between the two centres concerning the oral manifestations of diseases of the blood-system. As a result, I have had access to clinical material which may not otherwise have been seen so early; in return, I hope that some of the diagnostic facilities we are able to offer have proved of practical value to the dental clinician and that the active therapeutic advantages we provide for certain hæmorrhagic blood dyscrasias have simplified his task.

Practical experience of such a link with dental colleagues has demonstrated this value on several occasions where an astute observation by the dentist has resulted in a patient receiving rapid benefit from the early diagnosis and treatment of a general medical condition. Indeed, the diagnostic opportunities are so numerous that I hope this discussion may underline some of the points where we can be of mutual assistance.

Of the patients we see in this context there are those with blood-disorders which lead to oral disease or bleeding, and, on the other hand, to balance matters, there is an increasing number of instances where the hæmatologist requires advice and help on the dental management of his own cases. These patients may be grouped into four categories:—

1. Those, often with a previous history, who suffer immediate or delayed post-extraction bleeding, with no apparent local cause.

2. Those, usually with gingival pathology, referred for periodontal advice and found to present some readily recognizable medical condition. Those attending for routine dental supervision when the observant clinician notes a coincidental lesion.

3. Dental conditions for which hæmatological investigation offers diagnostic help: Hæmolytic disease of the newborn; Porphyria; Heavy metal poisoning; Hereditary hæmoglobinopathies; Genetic studies involving blood-group analysis.

4. Patients receiving treatment from a hæmatologist, where either the disease chronicity or its therapy may lead to dental complications.

With these headings in mind, I would like now to discuss some of the problems in greater detail. First, cases with post-extraction bleeding; I refer, of course, only to those where no local explanation is forthcoming. Some time ago I was asked whether a substantial proportion of such patients might suffer undiagnosed liver dysfunction with depression of prothrombin activity leading to poor clot formation. If such people existed, the routine oral use of vitamin K analogues might serve to reduce bleeding. Accordingly, we undertook the screening of clotting mechanisms in those patients referred to us in whom no local cause could be demonstrated. Such investigations as are available in our present state of knowledge of coagulation are tedious, but we have now examined 136 patients. The majority were referred from the out-patient service of the Royal Dental Hospital, and I am very grateful to my colleagues for their co-operation. These patients represent a highly selected group, for many were referred in the first instance by dental practitioners and only those in whom local measures proved ineffectual were passed on to my department: hence the relative incidence of disorders diagnosed has no significance.

Table I shows that there was no sex differentiation. In roughly one-third no explanation could be offered; many of these had a clear previous history and some had a vague family history of bleeding or easy bruising.

* Presented to the meeting of the British Society of Periodon tology held on February 8, 1960.

Failure to provide an explanation is thus an indication of the limits of our diagnostic ability.

Table I.—Post-extraction Bleeding

Abnormality detected	85	(men 38; women 47)
No abnormality found	51	(men 27; women 24)
Total	136	(men 65; women 71)

Of those found to have some demonstrable anomaly (*Table II*), roughly one-third were found to suffer an inherited defect, either of coagulation or of vascular integrity: due to sex linkage, the majority were males. In another third, the bleeding was directly referable to severe iron deficiency, usually in women. Finally, a third of those found to be abnormal had increased vascular fragility (often women with no family history to suggest a genetic fault) or a minor deviation from normal of prothrombin function, insufficient to incriminate liver disease or any recognized disorder of coagulation and not responding to the administration of vitamin K_1.

Investigation of this series has not provided any useful positive information, but it demonstrates clearly that, in a sizeable group of patients referred for post-extraction bleeding, occult liver disease plays no discernible part. Only one patient had liver disease; this was clinically apparent and the bleeding tendency failed to respond to vitamin K_1 either orally or parenterally.

The three recognizable causes* leading significantly to post-extraction bleeding are thus:—

Inherited defects, usually males;

Unpredictable vascular fragility, usually females;

Unexpected iron deficiency, usually females.

This is no time for discussing the various mechanisms upon which blood-coagulation is dependent. We do not yet fully understand this complex series of reactions and such knowledge as we do possess could well form the basis of a separate discussion. We can say, however, that where diagnosis is possible it is essential for it to be made with confidence, as some of the more common inherited defects can now be controlled for periods long enough to permit surgical intervention. Hæmophilia is no longer such a problem as to justify the retention of badly carious teeth.

I would like to stress one simple factor which is frequently overlooked, particularly in treating patients in whom an inherited anomaly is already known to be present. Spontaneous bleeding or its corollary, spontaneous coagulation, is dependent upon the simultaneous integrity of three factors—the vessel wall, platelet efficiency, and the presence in equilibrium of the soluble protein fractions (*Fig.* 1). Except as a result of direct damage, spontaneous bleeding will not occur unless *two* or more of these are defective.

Table II.—Post-extraction Bleeding—Abnormal Hæmatology

	Men	*Women*	*Total*
I. Inherited defects:			
Clotting factors	**18**	3	
Platelets	1	4	29
Vascular fragility	1	2	
II. Vascular fragility without family history	4	**17**	21
III. Abnormal prothrombin consumption or slight delayed activity	8	4	12
IV. Symptomatic:			
Anæmia	—	**11**	
Polycythæmia	4	2	23
Thrombocytopenia	1	4	
Liver disease	1	—	
		Total	85

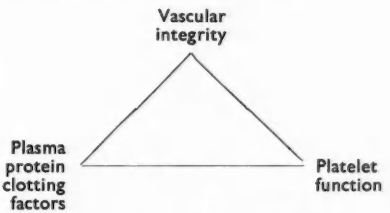


Fig. 1.—Spontaneous coagulation.

Following dental extraction, of course, only one other failure is required. The presence, for example, of hæmophilia does not preclude other pathology, and it is always worth investigating further if *spontaneous* purpura occurs in such patients: the second causal element may be amenable to treatment. Another example is the bleeding or bruising found in polycythæmia rubra vera, where coagulation

* The relevant numbers are delineated in bold type in *Table II*.

defects due to platelet instability may co-exist with vascular damage due to venous engorgement, stagnation, and anoxia. To carry this to its logical conclusion, it is always worth while giving fresh blood containing intact platelets to any serious bleeding dyscrasia, even if the diagnosis is obscure. By such empiricism and the use of supportive preparations such as vitamins K_1 or C, one of the two underlying failures may be corrected.

The second group of patients referred for hæmatological review comprise those suffering from diseases in which bleeding may often occur but is not a necessary feature. Clinical hæmatology relies to a great extent on the diagnostic value of changes in the oral mucosa, and the assiduous dental clinician has much scope for exercising the art of differential diagnosis!

I cannot hope to cover this field, but my own experience may be of some interest for I have now seen over 40 patients, other than those with post-extraction bleeding, referred from dental surgery because of some astute observation. Usually the patient is accompanied by the correct diagnosis, but occasionally we are able to bolster our own ego by revealing the unexpected. Conditions such as pernicious anæmia, cyclical neutropenia, hereditary telangiectasia, and porphyria, readily lend themselves to recognition; some, like pregnancy, measles, and early glandular fever, are more esoteric! Leukæmia and iron deficiency are hardy perennials, and the most protean in their manifestations.

Table III.—Causes of Glossitis, Mucosal Ulceration, or Buccal Purpura

1. Inherited clotting or vascular defects
2. Loss of epithelial integrity
   - —Enzyme deficiency
     - (iron)
     - (malabsorption syndrome)
   - —Defence failure
     - (leucocyte changes)
     - (antibody deficiency syndrome)
     - (iatrogenic)
3. Acquired clotting or vascular defects

Excluding the exanthemata, allergies, and endocrine disorders, in each of which blood-examination may be diagnostic, the majority of patients attend with either glossitis, mucosal atrophy or ulceration, or purpura. To put these symptoms into some perspective, I have tried to categorize them under three main aetiologic headings but, like many attempts at simple classification, this is arbitrary and contradictory (*Table III*).

I would like to take these briefly in order. First, inherited anomalies, other than those due to a protein factor deficiency such as hæmophilia, are more readily recognized and less uncommon than many appreciate. Familial deficiencies or functional defects of platelets may occur; the latter is known as Glanzmann's disease. Nothing can be done about these conditions, but *spontaneous* hæmorrhage almost certainly means some coincidental vascular change which may respond to simple remedies like vitamin C: bleeding can be controlled locally by fibrin foam and accurately applied pressure. A second category receiving attention these days is the familial vascular defect known as *pseudo*-hæmophilia or von Willebrand's disease, in which some ill-understood defect of the capillary wall leads to a prolonged bleeding time and spontaneous hæmorrhage. It is fascinating to find that many of these patients have a coincidental, often minimal, deficiency of anti-hæmophilic globulin, and the value of diagnosis in such cases lies in the fact that the anti-hæmophilic globulin deficiency can be temporarily corrected. A second curious feature of this anomaly is the waxing and waning of the clinical severity of its expression so that there is a real value in repeated assessment of the bleeding time to discover the optimum moment for surgical intervention.

The hereditary form of multiple telangiectasia (Osler–Rendu), a simple Mendelian dominant, usually may not become clinically manifest until the third or fourth decade, and then can be recognized early by characteristic small capillary dilatations on the buccal aspects of the lips and under-surface of the tongue. Early diagnosis is of scant help to the patient except to explain an otherwise obscure epistaxis or a chronic iron deficiency state, for the oral lesions usually presage similar changes scattered throughout the gut wall.

The second group comprises the majority of patients referred for reasons other than post-extraction hæmorrhage. They have lesions resulting from the loss of mucosal integrity. This epithelial failure leading to atrophy, glossitis, or ulceration, and even to purpura, has many causes with little or no basis in common.

If I may digress for a moment, I would like to emphasize that we are breaking away from the older holistic conception of one disease, one cause. Study of oral pathology offers an excellent opportunity to judge the other factors which play a part; no body surface is more regularly insulted than the buccal mucosa, and functional failure may equally well be due to exogenous environmental factors or to some genetically determined susceptibility. The so-called aetiological agent serves merely as a trigger mechanism.

Take one of the commonest examples, that of glossitis due to iron deficiency. The Plummer–Vinson syndrome associating this with dysphagia and koilonychia was conceived to be due to the hæmoglobin deficiency and local anoxia of the anæmic state. We now suspect that local mucosal integrity is hazarded if iron-dependent intracellular enzyme systems become blocked. Undoubtedly dysfunction is made worse if tissue anoxia co-exists, but it is the more subtle influence of failure of cytochrome oxidase systems which leads to the clinical symptom of sore tongue.

I have, therefore, stressed the question of enzyme deficiency acting locally as a significant cause for the disease becoming manifest. Of course, in the case of iron deficiency, treatment which replaces iron in the body will correct both the local enzyme defect and the hæmoglobin at the same time—it is scarcely surprising that the one has always been connected with the other. Recently I had referred to me a patient with mouth ulcers and a normal hæmoglobin concentration, but a significantly low serum iron level. Treatment with parenteral iron almost immediately cured the condition which had been present for seven years.

By malabsorption syndromes, we now refer to conditions in which absorption of vital dietary factors becomes impaired either from inherited defects (B_{12} deficiency in pernicious anæmia); interference with small-intestinal absorption (folic acid deficiency following surgical short circuits; bacterial competition and the blind loop syndrome; physiological changes in pregnancy; idiopathic steatorrhœa). Failure to absorb such entities and, to a lesser extent nicotinic acid, riboflavin, pyridoxine, and ascorbic acid, leads to enzyme deficiency which may block the formation of blood-cells with resultant anæmia, or may interfere with epithelial function, causing, in the mouth, mucosal ulceration or glossitis.

The interest in studying such malabsorption syndromes does not lie only in the precise medical conditions I have described. As age advances, physiological efficiency becomes reduced and some of these enzyme defects may become apparent with no other medical evidence except glossitis: a classical example of this is the sore tongue and glossal atrophy so often associated with dyspepsia in the elderly. This frequently responds to topical B_{12} in the form of a mouthwash and often is improved by simple iron therapy.

Although it is so very useful for a dental surgeon to diagnose pernicious anæmia as a result of observation, opportunities for this are rare. Yet glossitis among the older age groups is relatively common and often amenable to simple treatment. If this is true of changes in the tongue, it must equally be true of changes in the gingival tissues.

Loss of epithelial integrity may also signify failure of natural defence processes necessary for mucosal maintenance. Changes in white-cell activity are the commonest and are usually very well known and easily recognized. Agranulocytic angina does not need to be described. Once again, however, I feel it is useful to stress that minor changes may occur with a reduction of mucosal efficiency and a susceptibility to disease, where an early blood-count may demonstrate the cause before the florid picture of angina becomes obvious. Cyclical neutropenia is a fascinating example, although rarely recognized. We have been following a case, referred from the Royal Dental Hospital, Leicester Square, of a young

man who regularly shows an absolute and almost complete neutropenia about every three or four weeks; for this we have no explanation, but we assume that it is associated with some endocrine imbalance whereby the natural stimuli for white-cell production fail to evoke a response. Another example of this type is the neutropenia associated with enlargement of the spleen. Although rarely seen as an extreme lesion, it is not uncommon, particularly in association with rheumatoid arthritis, in which cases the spleen sooner or later enlarges. Occasionally, splenectomy may provide a complete cure of the recurrent buccal lesions which can be so irritating to the patient—it is worth remembering that rheumatoid arthritis is treated by drugs such as phenyl butazone, which are themselves capable of causing neutropenia.

The outstanding leucocyte changes which we see are referable to primary bone-marrow disease and, usually, myelogenous leukæmia. To-day, some 50 per cent of leukæmia is diagnosed in the presence of a normal or subnormal circulating white-cell level, and it is not possible simply to equate the white cell-count to the oral lesions. Once again, one might invoke as an explanation the local deficiency of enzyme response for mucosal repair, in this case provided by functioning leucocytes. It is known in leukæmia that intracellular enzymes become abnormal even though the white cell-count remains low; for example, alkaline phosphatase activity is invariably low in the cells of patients in whom glossitis and buccal ulceration show as marked symptoms. Later in the disease concomitant anæmia and thrombocytopenia will exaggerate superficial changes and the final clinical outcome results necessarily in a combination of all these defects. We do not know why the monocytic variant of myeloid leukæmia presents so classically with changes in the gums; it can be of real diagnostic significance, for one rarely sees comparable changes so early in the other forms of acute leukæmia, particularly in adolescents and young adults.

Monocytic leukæmia, which is referred to under the eponym of Naegeli, is a curio in other respects. It occurs in some 5 per cent of acute leukæmias and, as it is usually found in young adults, it is one of the more rapidly fatal lesions to deal with. In the early stages, its diagnosis is difficult, for typically there is only a slight increase in circulating white cells and, as these are very similar to the atypical Downie cells, it is always a problem to differentiate this condition from early acute glandular fever. Indeed, there may be some subtle connexion between the two, for the Paul Bunnell test, which is otherwise so specific for the latter, may occasionally show a false positive in this type of leukæmia. The one cardinal distinction between the two conditions is the invariable and progressive anæmia which always accompanies acute failure of the bone-marrow.

I would like to talk more on the problem of leukæmia, in which leucocyte changes lead to mucosal disease, but I wish to do so under another heading. In the maintenance of chronic leukæmia, and during therapy of acute leukæmia, mucosal changes occur which are more probably ascribable to the activities of the physician and I shall therefore refer to them as iatrogenic. Before discussing these, there is one other category of defence failure which is relevant, and I refer to the so-called antibody deficiency syndrome. This is a title which many of us prefer, but it has been more completely described as either congenital or acquired agammaglobulinæmia. In this condition, failure to form antibodies results in defective defence against disease, and this is shown clinically by recurrent attacks of trivial infections, many of which assume a severity which is out of proportion. In its grossest form, the disease is easily recognized, for the patient is able to survive only by virtue of antibiotic screening. We now know, however, that there are many intermediate grades, where the inability to form antibodies is not absolute, but where an insufficient concentration leads to recurrent but modified infection. In such patients, low-grade sepsis in the mouth, recurrent infections of the gums, and a retardation of healing after extraction may provide a clue as to the underlying cause. In the mild cases treatment is certainly not indicated and such patients often grow out of

this disability. In more severe cases, however, gammaglobulin can be given intravenously for periods long enough to permit the healing of superficial ulceration.

I would like to devote a few minutes to a discussion of diseases in the mouth due to the therapy of the physician. There are many causes, some of which you know full well—the agranulocytosis associated with gold therapy, anti-thyroid drugs and, more recently, phenyl-butazone are well known. To this may be added the reaction of individuals to almost any drug, where there is a pre-existing and possibly inherited susceptibility because of intracellular enzyme defects. Such cases have hitherto been attributed solely to the proprietary preparation; this serves as a trigger, but in many instances it is fairer to regard the patient as being equally at fault. A noteworthy example of such a state of affairs is the acute hæmolytic anæmia brought about by ingestion of the fava bean, or the inhalation of its pollen; it is now known that those susceptible to this disease suffer an inherited enzyme defect of the red cell whereby glutathione stability is impaired.

I do not wish, however, to discuss these but rather three types of oral lesion which we are now beginning to see as a result of modern therapeutic measures. There is a further type which probably results from prolongation of human life in diseases where death would formerly have shortened the natural history and excluded other changes (*Table IV*).

Table IV.—Iatrogenic Blood Dyscrasias
Drug sensitivity and toxicity
Alkylating and antimetabolite therapy
Prolongation of life in malignant disease
Anticoagulant therapy

To take the latter first, I refer to prolonged survival in the chronic leukæmias and malignant reticuloses as a result of conservative measures and the use of chemotherapy. In these cases, and classically this is found in chronic lymphatic leukæmia, the deranged reticulo-endothelial system begins to form abnormal antibodies, some of which are directed against the patient's own blood-cells. This so-called auto-immune disease may lead to hæmolytic anæmia, neutropenia, and even a drop in circulating platelets with purpura. *Pari passu* with this, the spleen enlarges and the syndrome known as hypersplenism also contributes to these changes. As a consequence, such patients tend to complain of buccal ulceration, glossitis, purpura, and bleeding, particularly under dentures. These changes rarely appeared before because patients died in an earlier phase of the disease, hence the occurrence of this symptom-complex is a price paid for success in lengthening the survival time of the individual. I now take great care in instructing patients about oral hygiene and, as the majority are elderly, I ask my dental colleagues to check the fitting of dentures which are often loose as a result of tissue resorption.

Another iatrogenic disease in the mouth is a very troublesome and disconcerting buccal ulceration which is the direct result of either alkylating or antimetabolite therapy. Antimetabolites and antivitamins are compounds which are strictly similar to metabolites and vitamins of physiologic importance; because of this similarity they are able to compete with the vitamin or metabolite concerned and, being inert, they block function. In the control of leukæmia this form of therapy is common, for it is used to interfere with the metabolism of developing white cells and hence to a reduction of the marrow output. Not only do such drugs reduce white-cell activity, but they will also interfere with intracellular enzyme function throughout the body. In acute leukæmia of bone-marrow origin, the antivitamin aminopterin and its derivatives are used to compete with folic acid and hence to interfere with nucleic acid synthesis. These folic acid antagonists sooner or later lead to buccal ulceration which can be very painful, and any oral sepsis or dental pathology is liable to be exacerbated; the problem is difficult, for this drug is used largely in the treatment of leukæmia in childhood.

The antimetabolite 6-mercaptopurine competes with purine metabolism somewhat later in the same nucleic acid cycle. It is used widely in the treatment of acute leukæmia at all ages, and, although its side effects are

considerably less, its prolonged use again tends to result in painful lesions in the mouth.

These two types of drug are used for the attempted control of leukæmia. Another form of chemotherapy available depends upon cytotoxic effects, whereby cellular mitosis is interrupted, and this class of drug, usually a derivative of nitrogen mustard, acts by what is termed alkylation. Alkylating drugs are being used with success in the long-term therapy of chronic leukæmia, and, more recently, their use has been extended to the control of disseminated malignant disease. At the therapeutic range, nitrogen mustard derivatives do not usually damage the mouth, although this may happen in isolated instances; we are now entering a phase in which very considerably increased doses are given, leading to almost total destruction of the bone-marrow as well as of the neoplastic or leukæmic process.

This advance in alkylating therapy has become possible as a result of recent measures introduced, which permit the low-temperature storage of bone-marrow taken from the patient before therapy and reintroduced after the immediate therapeutic hazards have passed. The success of this approach, whereby much larger doses can be given without fear of permanent marrow ablation, has resulted in bodily changes not hitherto experienced. After bone-marrow, the next most sensitive structure appears to be the epithelial lining of the intestinal tract; patients now receiving high dose alkylating therapy are prone to show damage to the buccal mucosa, partly because the circulating level of white cells is depressed almost to extinction, and partly because of the direct effect of the drug on the metabolism of the epithelial cell itself.

We believe that this form of therapy is going to become more popular and will be continued on a maintenance basis for months and even perhaps for years. We therefore predict that patients suffering malignant conditions will begin to present problems in the mouth similar to those seen in the chronic leukæmias.

There is one final therapeutic measure which may result in changes involving the dental surgeon. I refer to the astonishing rise in out-patient anticoagulant therapy, mainly for those suffering from ischæmic heart disease. At St. George's Hospital, over the past four years, some four hundred out-patients have been treated in this way, and we are concerned at the speed with which this out-patient clinic is growing. On the whole, we advise such patients to consult the dental out-patient department for extractions because we believe it is possible, and indeed preferable, to carry out this treatment without modifying the anticoagulant regimen.

It has been assessed, rather pessimistically, that there are some 500,000 people who might logically receive this form of treatment in England and Wales. Accordingly, general dental practitioners are going to become increasingly involved. This is really a subject for a separate discussion and I can do no more than refer to it in general terms, but I might mention that the depression of coagulation factors should not preclude extractions, unless of an extensive nature; current anticoagulant therapy does not prevent coagulation, but merely delays it, and the final fibrin clot, once formed, is normal.

There only remains for me to discuss the class of patient with mucosal lesions resulting from acquired clotting and vascular defects. Once again, some of these can be recognized at an early stage and many are symptomatic of a systemic medical condition. Most are well known, but I would like to refer briefly to acquired platelet deficiency. Here again is an instance of auto-immune disease. Many of the cases hitherto recorded as idiopathic thrombocytopenia purpura are now understood to result from the circulation of a platelet antibody leading to agglutination and destruction of platelets in the spleen. Occasionally such cases are first seen following dental extraction; many spontaneously remit and others respond to corticosteroid therapy, but between 10 per cent and 20 per cent may become chronic. Of some interest in this group is the occasional case in which formation of the anti-platelet antibody is in some way provoked by the association or complexing of

certain drugs with the platelets. Some apparently innocuous preparation such as the sedative apronal, or the cardiac depressant quinidine, can lead to this phenomenon and, in one instance, I have seen it occur following the routine administration of an oral antibiotic given to clear the mouth prior to multiple extractions.

Another acquired vascular deficiency of mounting clinical significance is the telangiectasia or vascular dilatation and fragility seen in association with senility. There must be many causes, but basically old age leads to loss of supportive tissue around a vessel and bleeding occurs readily. We now find that this condition, essentially a physiological failure, is significantly allied to iron-deficiency anæmia in old age. Many old ladies with low-grade chronic anæmia and a small amount of occult blood in the stools present with no other pathology. Careful observation in the mouth will often show the telangiectasia, in this case acquired, which may provide the diagnostic clue. By itself, this condition is innocuous, but it is so frequently coupled with symptomless hiatus hernia which with gastric congestion leads to constant and insidious bleeding. We now appreciate that the majority of elderly patients, presenting with iron-deficiency anæmia for the first time, suffer from this to a greater or lesser extent.

These then are some of the features of oral pathology with which the hæmatologist and the dental surgeon are mutually concerned. I have tried to demonstrate where, in some instances, the laboratory can assist diagnosis and treatment, in other cases where the dental practitioner may be the first to draw attention to a significant diagnostic feature and, finally, to an increasing number of patients for whom the hæmatologist requires the advice and co-operation of the dental clinician.

BOOK REVIEWS

TRAUMATISMES ACCIDENTELS EN STOMATOLOGIE. Massif Facial et Incisives Supérieurs. By P. MARONNEAUD, M. PERI, C. CREPY, and J. LACHARD. Report presented at the 16th Congrès Français de Stomatologie, Paris, October 5–10, 1959. $9\frac{1}{2} \times 6\frac{1}{4}$ in. Pp. 292, with 43 illus. 1959. Paris: Masson et Cie. Paper covers.

THIS report was presented at the sixteenth French Congress of Stomatology in Paris, October 5–10, 1959.

The authors have gone to considerable trouble to present their subject very clearly and in great detail. They point out that with the increase of motor transport, accidents occur very much more frequently and a large proportion involve the facial skeleton and the upper front teeth.

They state with justification that road accidents are the scourge of modern times. Vehicles with two wheels are involved in more accidents than those with four. They show from their investigations that the severity of the injuries often depend on the position the person is actually occupying in the car. Children are less likely to sustain fractures owing to the elasticity of their bones. They go into considerable detail with the various types of fractures. It is interesting to note that the comparatively strong malar bones often take up the first impact of the shock, and so protect the thinner and weaker parts of the facial skeleton such as the orbit, nasal passage, and sinuses. The book could be improved with more diagrams which would explain more than some of the over-detailed descriptions.

Methods of treatment such as fixation by splints and plaster headcap, packing with gauze, and intra-osseous wiring are all fully described; they rightly stress the importance of early treatment whenever possible; delay should only be allowed when the general condition of the patient does not permit of prompt intervention.

The need for antibiotic treatment in the complicated injuries to the face is rightly stressed and also the use of endotracheal anæsthesia. The authors have found, as have operators elsewhere, that the only effective

way to treat such injuries is at a special centre by a team used to the various specialities necessary to produce full treatment.

The upper incisor teeth being prominent often take the first shock of any force in road accidents, frequently being damaged, or knocked out completely.

The various degrees of injury are discussed and treatment by restoration, prostheses, and re-implantation of the teeth are all duly considered. Anyone interested in this type of work would be well rewarded by reading it, as the authors certainly have gone to considerable trouble to elucidate their subject.

G. L.

THE RESILIENCY OF THE EDENTULOUS ALVEOLAR RIDGES. By THOR ARSTAD, Professor of Prosthetic Dentistry at the Odontological Faculty of the University of Oslo. 9×6 in. Pp. 90, with 33 illus. 1959. Oslo: Oslo University Press (London: Wm. Dawson and Sons Ltd.).

THE registration and recording of mandibular positions and movements have always presented a problem to the full denture prosthetist. When to this problem is added that of movable bases on resilient tissues, it is perhaps not surprising that the majority of dentists resort to the so-called plane line articulator as a protest against so many possible errors.

In a monograph on the relationship between these two problems, Professor Arstad examines the difficulties and reports on the results of two series of investigations he carried out. The first was conducted so that discrepancies in occlusion of the teeth could be calculated when the movements of the mandible deviated from those in the articulator. As a result he was able to calculate the displacement of the dentures necessary to produce an even contact between the teeth. In an appendix, the methods for making these calculations are presented and will provide an enjoyable hour for those who remember their trigonometry. In the second series, the author examined various known methods of registering mandibular movements in an attempt to assess their accuracy. He is unable to guarantee a correct registration with any of the methods used, but observes that occlusion in the mouth was better than might be expected due, he claims, to the property of resiliency of the underlying mucosa. This is not intended to provide a crumb of comfort for the user of the plane line articulator but rather for those who prefer the average value instrument to the more complex one.

At times the text makes difficult reading since three-dimensional thinking is required, but it is a piece of work worthy of study. By way of criticism, there is occasional confusion over the use of words such as compression and displacement which are sometimes used synonymously. Also there are but three scant lines in the text devoted to the histological structures of the tissues discussed. But, in general, this monograph will prove of great value to serious prosthetists and a stimulus to those who would be more serious.

H. T.

Degree of Keratinization and Glycogen Content in the Uninflamed and Inflamed Gingiva and Alveolar Mucosa

A study of the degree of inflammation, keratinization, and glycogen content in bilateral biopsies of gingiva and alveolar mucosa obtained from 52 men in the age-group 27–70 years revealed four types of keratinization and variable amounts of glycogen in both the inflamed and uninflamed gingiva.

In the absence of inflammation it was found that the degree of keratinization decreased from the attached gingiva towards the crest, whereas glycogen was more apparent in the crest and less marked in the attached gingiva.

In inflamed regions keratinization was reduced and glycogen deposition increased.

The epithelium of the alveolar mucosa was non-keratinized and contained glycogen in all specimens.

Keratinization and glycogen content are inversely related in uninflamed gingival regions, more markedly in inflamed gingival regions, and most markedly in the alveolar mucosa.—WEISS, M. D., WEINMANN, J. P., and MEYER, J. (1959), *J. Periodont.*, **30**, 208.

FURTHER REPORT ON A CASE DEMONSTRATING BODILY MOVEMENT OF UPPER INCISOR TEETH

By **J. R. HALDEN**, L.D.S. R.C.S., B.D.S., D.Orth. R.C.S.

EARLIER this year Dr. A. Goldstein of Chicago gave a paper at Guy's Hospital in which he showed a number of treated cases which had relapsed, especially in the sense that they had reverted to their former facial pattern. The inference was that it is not worth while to attempt to improve facial æsthetics by advanced treatment procedures because they are doomed to relapse anyway. Instead of occasioning sadness this viewpoint was received with such great glee in some academic quarters that I wondered whether there was not a little malicious joy on the part of those who are reluctant to believe that these advanced procedures can be successful. Time alone will tell, but meantime I thought you might like to follow up one case that I reported on in my paper on the edgewise appliance. I will repeat what I said then:—

Fig. 1.—Photographs at commencement of treatment.

"This girl, aged twelve years two months, was an Angle Class I, mild Skeletal II with increased overbite and slightly increased overjet associated with slight lip incompetence at rest (*Fig.* 1). I think her mandible is a bit depressed from true rest position in the lower pair of photographs. *Fig.* 2 shows the models before treatment. It was felt that she was perhaps a little too full around the lips, and that her profile might be improved and her lips made competent by retraction of the lower

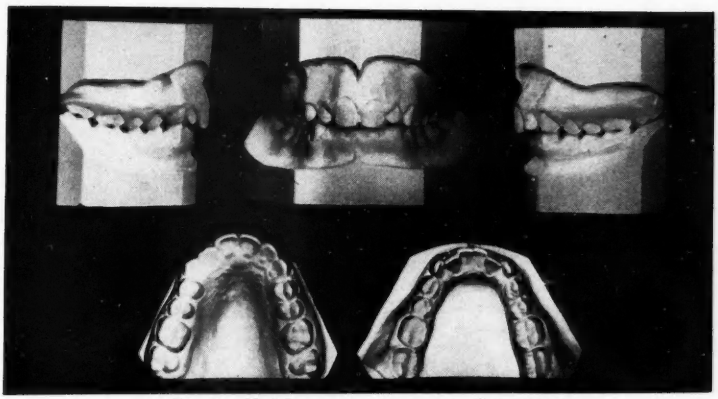


Fig. 2.—Models at commencement of treatment.

Given at the meeting held on December 14, 1959.

labial segment to about the position where it was eventually taken, followed by bodily retraction of the upper labial segment to the lower labial segment. Four first premolars were extracted, and treatment basically as outlined by Tweed carried out. *Fig.* 3 shows the models at retention. The headplates (*Fig.* 4) show that there has been a considerable reduction of dento-alveolar convexity, and that the upper incisor apices are nearer the palatal wall after treatment. The tracings (*Fig.* 5), super-imposed on SN and Sella, show that the upper central incisor crowns were retracted 8 mm. and their apices were retracted 6 mm.,

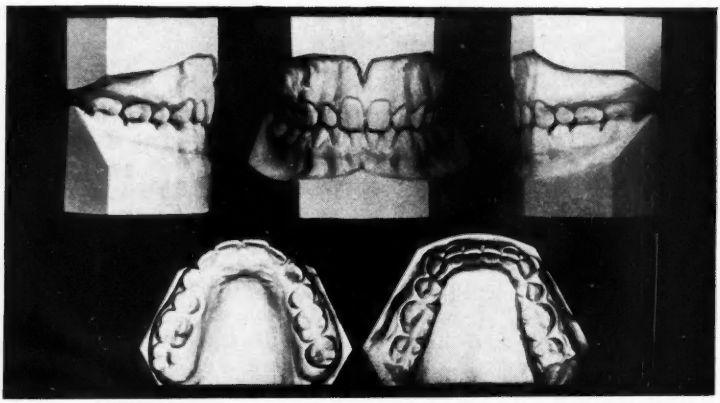
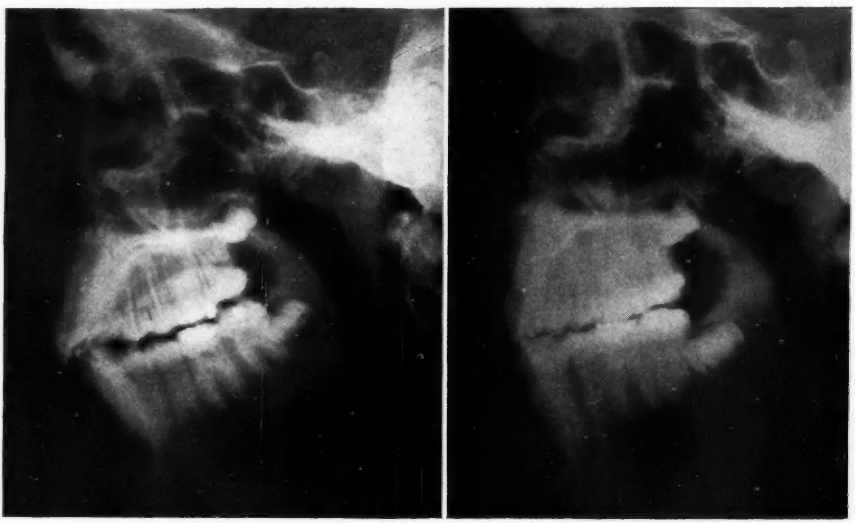


Fig. 3.—Models at retention.



A B

Fig. 4.—Headplates before treatment and at retention. Mandible postured forward a little in A. Note upper incisor apical position relative to A point and palatal wall before and after treatment. The bone anterior to upper incisor apices in B will disappear with a consequent posterior shift of A point.

less a small magnification correction for the conventional 5 feet target distance. The photographs after treatment show that her lips became quite competent, and the profile perhaps a little too flat for some tastes (*Fig.* 6);

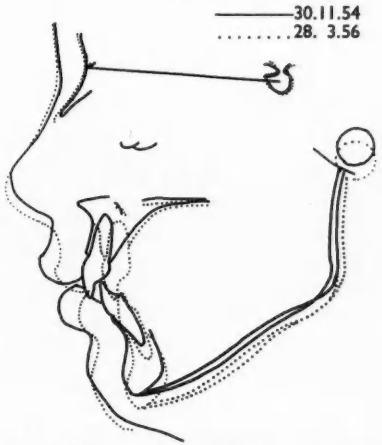


Fig. 5.—Tracings before and after treatment.

This girl was the first of the eight cases I showed to come out of retention because she broke three upper and three lower retainers and I refused to make any more. She had just over thirteen months' retention instead of the two years I usually require. At that time I thought it was good treatment to allow some expansion of the lower canines during their retraction, provided they were kept within the line of the arch. I was wrong. Nearly all those

Fig. 6.—Photographs at retention. Optimum change is still to occur when occlusion has settled in.

Fig. 7.—Models nineteen months out of retention.

however, I am nearly certain that when her occlusion settles down after retaining plates are left out she will show an optimum facial change in her morphology. Treatment time from extractions to retention was fifteen months."

cases have relapsed to the extent that the lower canines have reverted to their former width across the median plane, often to less than their former width.

These models (*Fig.* 7) were made nineteen months after retention was discontinued.

Fig. 8.—Headplate taken seven months out of retention. Note that the bone mesial to the upper incisor apices has disappeared and that the apices are still back relative to the palatal wall.

Fig. 9.—Headplate taken twenty-seven months out of retention. Note no change in incisor position has occurred in twenty months.

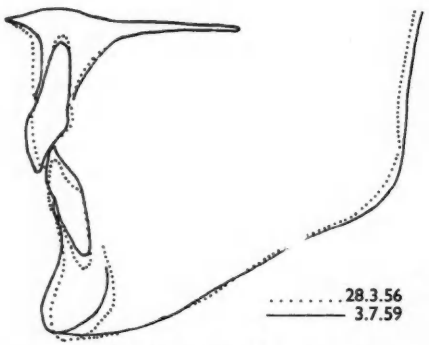


Fig. 10.—Tracing of headplate twenty-seven months out of retention superimposed on tracing of headplate at retention.

Fig. 11.—Photographs before treatment and two years out of retention.

Lower canine width is now less than the original width, which isn't fair!

Fig. 8 shows the position seven months only after retention was discontinued. Note that overbite is now normal but that the bone through which the upper incisor apices was moved has disappeared, and A point has been set back several millimetres. (*Compare Figs.* 8 and 4 B.) This bears out exactly what Holdaway and others have reported. Note that the upper incisor apices are still right back relative to the palatal wall.

Fig. 9 shows the position twenty months later, or twenty-seven months out of retention.

Note that there is virtually no change at all.

Fig. 10 shows the tracing of the slide just shown superimposed on the tracing of the headplate at retention, using anterior nasal spine as registration point.

Fig. 11 shows the adaptation of the facial contours to the new position of the denture and, despite the dark shadow, the refinement of the profile.

Acknowledgements.—I should like to thank Professor Ballard for permission to show this case, and the Photographic Department of the Eastman Dental Hospital for their preparation of the slides.

DISCUSSION

The President thanked Mr. Halden for his communication and hoped that others would bring forward similar reports so that developments in later life could be discussed.

Mr. Beresford thanked Mr. Halden for his interesting communication and hoped that he would be forgiven for asking an obvious question while the rest of the company marshalled their thoughts. Could Mr. Halden please tell us something about the vitality of these teeth? What was the effect if a light gauge round labial arch wire, or any similar apparatus, was used in an attempt to produce a similar tooth movement?

Mr. Halden replied that he did not know of the existence of any other apparatus which could be used.

CINÉRADIOGRAPHIC STUDIES OF TONGUE BEHAVIOUR

By **W. J. TULLEY**, B.D.S., F.D.S. R.C.S., D.Orth. R.C.S.
Guy's Hospital Dental School

THE advent of the electronic method of image intensification has enabled the radiologist to carry out prolonged screening of patients, and thus to observe movements of the soft tissues of the alimentary tract with the help of radio-opaque media. The X-ray dosage received by the patient is infinitesimal during such screening, and the quality of the picture is excellent.

This technique has, in recent years, been used to take ciné films of movements of the soft tissues in feeding and swallowing (Ardran and Kemp, 1952, 1955). More recently they have investigated the movements of the soft tissues in the suckling cycle (Ardran and Kemp, 1958).

This method of cinéfluorography has been used in the United States to study movements of the mandible and movements of the soft palate in cleft-palate patients (Cooper, 1956).

Grossman has shown films taken in this way to illustrate movements of the tongue and lips in Class II, division 2 malocclusions, and Leighton (1960) showed the use of this apparatus to study swallowing behaviour in infants.

This form of investigation is very relevant to our field of interest, but the technique is particularly difficult. In the lateral view, the teeth, because of their density, tend to mask the movements of the anterior part of the tongue so that the exposure of the film is a very critical matter (*Fig.* 1). Grossman tried to overcome this by placing a metal object on the tip of the lower incisor.

There is a danger of excessive X-ray dosage even with the image intensifier. In taking films with this type of apparatus the output of X-rays has to be increased considerably beyond that required for merely screening. This means that only a few seconds of film can be run and trial shots to perfect exposure cannot be made on the same patient. It is necessary to use another subject in the right age-group with similar facial characteristics, e.g., size of jaws and same density of soft tissues, to obtain a trial exposure.

Despite the present concern with the hazards of radiation, I have been assured that the few seconds of such filming are perfectly harmless, but there would be a danger in

Given at the meeting held on December 14, 1959.

making a serial study in children, as one would like to do.

At present this type of investigation cannot be carried out on a large scale to study the possible changes in tongue activity in the growing child. However, electronic equipment is being developed where films can be taken of television screens and the X-ray dosage is very small indeed.

Another problem is one of head fixation. It is extremely difficult to get a true lateral view of the younger child, and some form of cephalostat attached to the apparatus is desirable.

The film to be shown illustrates one example of what would appear to be the normal behaviour of the tongue and lips in swallowing and in speech. This is contrasted with a patient with a marked Skeletal 2 abnormality, Class II, division 1 malocclusion, a marked tongue thrust, and lisping speech. This type of tongue behaviour, taken in association with the skeletal abnormality, makes improvement of incisor position virtually impossible. This type of atypical behaviour was chosen because:—

1. The patient never made tooth contact at any time during swallowing.

2. The open bite and overjet enables the tip of the tongue to be more visible.

3. It is the type of innate tongue-behaviour which interests us most. (Rix, 1953; Gwynne-Evans, 1954; and Ballard, 1957.)

There are many imperfections in the film, and it is being shown at this stage for helpful

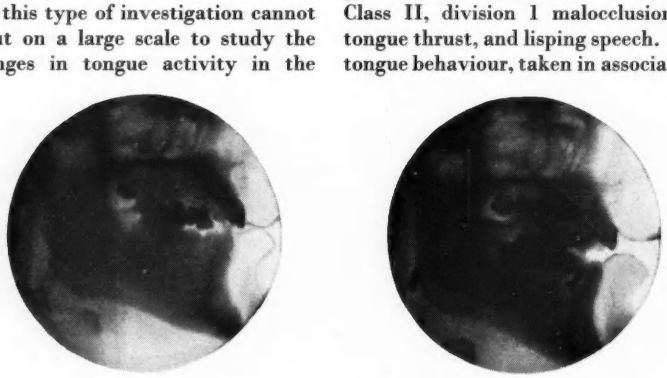


Fig. 1.—Extracted frames from ciné film showing the difficulties in exposure. A, Resting position of tongue. Outline of tongue and soft palate relatively clear. B, Tongue position in "s" sound. Tongue tip can be seen palatal to upper incisors. The full outline of the tongue is obscured by the density of the teeth and the barium which has leaked onto the side of the tongue.

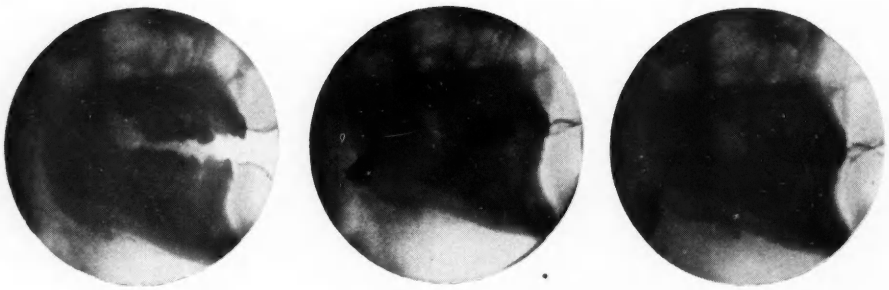


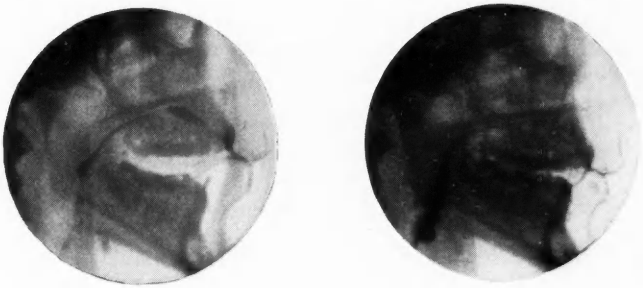
Fig. 2.—"Normal" swallowing sequence (patient with "normal" occlusion). A, Prior to swallowing with barium on dorsum of tongue. B, Teeth together at "mylohyoid phase". Tongue filling oral cavity. Barium spilling into mesopharynx-nasopharyngeal closure. C, Continuation of the last phase.

and constructive criticism. Apart from problems of correct exposure, different densities of barium paste were used to try to improve the soft tissue outline. The best results were obtained by mixing mucilage with the paste so that in certain parts of the film the soft tissues show poor definition whereas in others their outline is extremely clear. (Compare *Fig.* 1 A with *Fig.* 1 B.)

observing movements more closely. An animated viewer is the best way of making a detailed analysis of the movements, and this can be enhanced by tracings of individual frames.

The film was then shown with the following commentary:—

The first subject in this film is swallowing a small quantity of barium paste, performing



A B

Fig. 3.—"Abnormal" swallowing sequence (patient with Class II, division 1 malocclusion). A, Prior to swallowing with barium on dorsum of tongue. B, Mylohyoid phase of swallowing as barium is transferred into the mesopharynx. Note that the teeth are not together and the tongue protrudes between the upper and lower incisors in contact with the contracting lower lip.

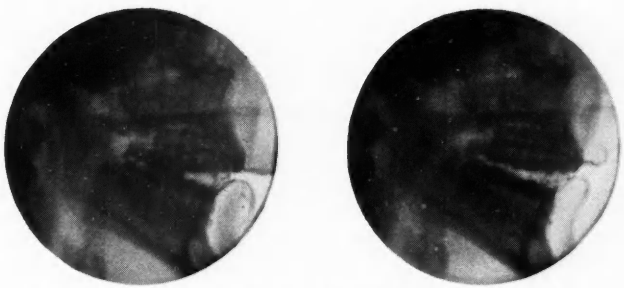


Fig. 4.—Interdental placing of tongue in "s" sounds. The tongue protrudes between the incisors to contact the lower lip.

The film was taken for me in the Department of Radiology, Guy's Hospital, using the Phillips Image Intensification apparatus. It was first filmed on 35-mm. film, from which extracted frames have been taken for prints and then reduced to 16 mm. for projection.

In order to increase the viewing time, the short series of film containing a few swallowing actions can be repeated several times for projection so that one has a chance of

what Rix (1946) has described as the basic swallow. You will note the way in which the liquid "bolus" is transferred to the back of the mouth by the tongue, as described by Whillis (1946), and at the mylohyoid phase of swallowing the teeth are placed together and the tongue, contained within the dental arches, bulges back so that the liquid is squirted into the mesopharynx. Nasopharyngeal closure and laryngeal protection is carried out in the

way so clearly described by Ardran and Kemp (*Fig.* 2).

As an example of speech the first subject says "sing a song of sixpence". In the "s" sounds the tip of the tongue is placed just behind the upper incisors and not between them (*Fig.* 1). The movements of the soft palate are clearly seen, and also the fact that the mandible is acting mainly by hinge mechanism during speech.

The second subject, Class II, division 1 with a Skeletal 2 dental base relationship, never occludes her teeth during any stage of swallowing. She has a gulping swallow as described by Rix and the tongue protrudes between the incisor teeth to make contact with the strongly contracting lower lip (*Fig.* 3). In speech she is saying "sing a song of sixpence", when the tip of the tongue is placed between the upper and lower incisors again in contact with the lower lip. Her mandible is protruded to compensate for the discrepancy in dental and jaw relationship, and at rest the tongue shows a forward position between the incisors (*Fig.* 4).

It was not the original intention to use this film for teaching purposes. It was to be merely an investigation of the possibilities of using this technique. It does, however, show graphically the atypical function of the soft tissues, and it was shown for a critical discussion on the technique.

Acknowledgements.—I would like to thank Dr. Dow and Mr. Stammers of the Department of Diagnostic Radiology, Guy's Hospital, for their co-operation and assistance in the preparation of this film, and Mr. Jones of the Dental Photographic Department for the preparation of the film for projection. I would especially like to thank Mr. Rix for his encouragement in this project.

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DISCUSSION

The President said that he thought they should thank not only Mr. Tulley but also the noble patients who participated in a remarkable and interesting set of films.

Opening the discussion, *Mr. Wilson* said that the standard of the presentation had been what they expected of Mr. Tulley, that is, very good.

In the second patient, there seemed to be a number of minor swallows where the tongue and soft palate did not make contact. Could Mr. Tulley explain this ? How was it proposed to treat the case?

Mr. Tulley replied that the movements of soft palate referred to had occurred when the patient was gagging. (*Laughter.*) It was a case which had received considerable orthodontic treatment and which had been referred for a second opinion. The film showed something of the soft tissues, but when the patient, a girl of 15, was examined clinically, her appearance did not seem to be too bad. Her mandible adopted a forward and downward postural position at rest and some conscious lip seal. It was felt that it was better to leave her alone.

Mr. Broadway congratulated Mr. Tulley and the Radiographic Department of Guy's Hospital on an excellent film. Did Mr. Tulley consider that the environment of the X-ray Department and the image intensifier and all the other apparatus had any influence on the swallowing mechanism?

Mr. Tulley said that Mr. Broadway had better ask one of the subjects of the film who was present at the meeting. He did not think that they were greatly influenced. The girl had produced the same sort of picture on clinical examination. He agreed that this method had very severe limitations in research work on that score and on the score of dosage. That was a pity because the method lent itself to serial examination.

Mr. Timms asked whether Mr. Tulley had any figures for dosage since they had found that 10–20 seconds of exposure was a radiation equivalent of less than one-half of one cephalometric headplate.

Mr. Tulley said that that was approximately what he had been told, but one was apt to get over-enthusiastic with the filming.

Mr. Timms said they had not been worried about that dosage. University College Hospital were now using a mixture of glycerin and tantalum powder instead of barium. It was very much easier to use and he thought that the patients liked it. The mixture gave no trouble and could be applied with a small paint brush.

Mr. Nicol said that Mr. Tulley might like to try using a mixture of malted milk with barium sulphide. That made a sticky mess and seemed to stick rather well.

Mr. Tulley said that he was sure that his subjects would be delighted to hear that.

The President asked if anyone else wished to put forward their personal recipe.

He said that he had found it a most interesting evening. They owed much to the hard work which the contributors had devoted to their papers, both in their preparation and presentation. He had to thank Mr. Tulley for his communication and he asked those present to show their appreciation in the usual way. (*Applause.*)